ORIGINAL ARTICLE

The Relative Benefits of Endurance and Strength Training on the Metabolic Factors and Muscle Function of People With Type 2 Diabetes Mellitus

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ABSTRACT. Cauza E, Hanusch-Enserer U, Strasser B, Ludvik B, Metz-Schimmerl S, Pacini G, Wagner O, Georg P, Prager R, Kostner K, Dunky A, Haber P. The relative benefits of endurance and strength training on the metabolic factors and muscle function of people with type 2 diabetes mellitus. Arch Phys Med Rehabil 2005;86:1527-33.

Objective: To compare the effects of a 4-month strength training (ST) versus aerobic endurance training (ET) program on metabolic control, muscle strength, and cardiovascular endurance in subjects with type 2 diabetes mellitus (T2D).

Design: Randomized controlled trial.

Setting: Large public tertiary hospital.

Participants: Twenty-two T2D participants (11 men, 11 women; mean age ± standard error, 56.2±1.1y; diabetes duration, 8.8±3.5y) were randomized into a 4-month ST program and 17 T2D participants (9 men, 8 women; mean age, 57.9±1.4y; diabetes duration, 9.2±1.7y) into a 4-month ET program.

Interventions: ST (up to 6 sets per muscle group per week) and ET (with an intensity of maximal oxygen consumption of 60% and a volume beginning at 15min and advancing to a maximum of 30min 3×/wk) for 4 months.

Main Outcome Measures: Laboratory tests included determinations of blood glucose, glycosylated hemoglobin (Hb A_{1c}), insulin, and lipid assays.

Results: A significant decline in Hb A_{1c} was only observed in the ST group (8.3%±1.7% to 7.1%±0.2%, $P=.001$). Blood glucose (204±16mg/dL to 147±8mg/dL, $P<.001$) and insulin resistance (9.11±1.51 to 7.15±1.15, $P=.04$) improved significantly in the ST group, whereas no significant changes were observed in the ET group. Baseline levels of total cholesterol (207±8mg/dL to 184±7mg/dL, $P<.001$), low-density lipoprotein cholesterol (120±8mg/dL to 106±8mg/dL, $P=.001$), and triglyceride levels (229±25mg/dL to 150±15mg/dL, $P=.001$) were significantly reduced and high-density lipoprotein cholesterol (43±3mg/dL to 48±2mg/dL, $P=.004$) was significantly increased in the ST group; in contrast, no such changes were seen in the ET group.

Conclusions: ST was more effective than ET in improving glycemic control. With the added advantage of an improved lipid profile, we conclude that ST may play an important role in the treatment of T2D.

Key Words: Hyperglycemia; Insulin resistance; Physical endurance; Rehabilitation.

THE EFFECTIVENESS OF physical exercise for the treatment of type 2 diabetes mellitus (T2D) has long been recognized.[1,2] Endurance training (ET) has been advocated as the most suitable form of exercise,[3,4] with many positive metabolic effects, such as improvements in lipid profile,[5] reduced body fat,[5] and decreased blood glucose (BG) levels.[5] ET also appears to be effective in improving insulin resistance in patients with T2D[6,7] and in obese subjects without diabetes.[8] By comparison, only limited information is available on the effect of strength training (ST) on T2D.[9-12] Reports on the effects of ST on glycemic control in patients with T2D have been controversial. For example, a 2-month trial with 11 patients with T2D reported that ST had no effect on glucose metabolism,[11] whereas another study[13] found only small improvements (0.5% difference in glycosylated hemoglobin [Hb A_{1c}] vs the control group) in patients with T2D after a 5-month resistance training program. In a third study,[12] 8 T2D patients who had participated in a 3-month circuit of progressive resistance training showed significant improvement ($P<.05$) in Hb A_{1c} that was associated with a significant increase in muscle tissue, as measured by magnetic resonance imaging. Two recent studies support the benefits of ST on glycemic control. First, Dunstan et al[10] reported a significant improvement of Hb A_{1c} (15%) after high-intensity resistance training in older T2D patients. After 6 months of resistance training in combination with a moderate weight loss diet, there was a 15% reduction in Hb A_{1c}. Second, Castaneda et al[9] showed improved metabolic control (Hb A_{1c} decreased from 8.7% to 7.6%, $P=.01$) by progressive resistance training in 31 Latino patients with T2D. Erikson et al[12] reported no significant changes in lipid levels with a moderate-intensity and high-volume resistance training program. Similarly, serum lipids and lipoproteins remained unchanged in the study of Dunstan,[10] whereas Castaneda[9] reported a trend toward a reduction in serum triglyceride (TG) levels within the progressive resistance training group compared with control subjects ($P=.08$).

One possible explanation of the positive effects of ST on insulin resistance (IR) may be the increase in the number of glucose transporter (GLUT) proteins. In skeletal muscle

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cells, GLUT4 is thought to be responsible for insulin- and contraction-stimulated glucose transport[14] in skeletal muscle. An increase in GLUT4 has also been observed after ST by Tabata et al.[15] In addition, increasing total muscle mass will ultimately result in an increase in total insulin-mediated glucose uptake. Another possible underlying mechanism for improved glucose uptake could be an increased number of insulin receptors in the muscle cell.

In contrast to ST, ET has different effects on skeletal muscles, the cardiovascular system, and the autonomic nervous system. ET increases skeletal muscle capillarization and blood flow, muscular GLUT4 levels, hexokinase, and glycogen synthase activities. In contrast to ST, the adaptations in skeletal muscle as a result of ET involve an increase in the capacity for aerobic metabolism made possible by an adaptive increase in mitochondrial content as well as a number of other enzymatic adaptations that may contribute to the altered metabolic response to exercise in the trained state.[16-18]

Abnormal insulin secretion, diminished glucose effectiveness, and both peripheral and hepatic IR are the primary pathogenic factors that lead to T2D,[6] which is a serious, chronic disease associated with hyperglycemia, obesity, and the metabolic syndrome.[19] In addition to obesity, hyperglycemia alone impairs insulin secretion and causes IR and thus makes the pathogenesis of T2D even more complex.[19,20] Hyperinsulinemia and IR are associated with several atherogenic changes that increase the risk of development of coronary heart disease.[21] These include dyslipidemia, especially abnormalities in total cholesterol (TC) with high levels of low-density lipoprotein cholesterol (LDL-C) and TG; obesity; and hypertension. Such abnormalities contribute to the risk of micro- and macrovascular complications.[6,21]

The skeletal muscle is responsible for up to 40% of total body weight. ST may induce beneficial changes in IR via muscle mass development. Skeletal muscle tissue is the major site of insulin-mediated glucose uptake and strongly influences IR, which is characterized by a decrease in glucose uptake into the skeletal muscle tissue in patients with T2D.[6] Because IR is an important modifiable risk factor for atherosclerosis, we studied the potential beneficial effects of ST versus ET on IR, muscular mass, and oxygen consumption ($\dot{V}O_2$) in patients with T2D.

METHODS

Study Population

We randomized 43 patients from our diabetes outpatient department—22 men (mean age ± standard error [SE], 56.5±0.9y; range, 51–69y) and 21 women (mean age, 57.4±0.9y; range, 50–70y)—between September 2000 and May 2002 who had T2D and no complications or comorbid conditions. The patients were consecutively divided into 2 groups (ST vs ET); none from either group was involved in organized ET programs. One subject did not complete the study because of health reasons unrelated to the investigation and 3 subjects did not complete the study because of private reasons. All participants had a fasting glucose concentration of 126mg/dL or greater (≥7.0mmol/L) and met the World Health Organization criteria for the diagnosis of T2D. Only patients between the ages of 50 and 70 years were accepted for the study. No limitations were given for body weight or body mass index (BMI). All demographic data are shown in table 1.

A physician performed physical examinations on all subjects before the study. Subjects were excluded if they had rapidly progressive or terminal illness, myocardial infarction, uncontrolled arrhythmias, third-degree heart blockage, elevated blood pressure (>200/100mmHg on therapy), nephropathy (microalbuminuria >20μg/min albumin excretion), severe peripheral or autonomic neuropathy, or diabetic proliferative retinopathy. Other exclusion criteria were severe musculoskeletal and neurologic abnormalities. Mild peripheral neuropathy was not considered a contraindication.

Table 1: Subject Characteristics and Treatment Regimens at Baseline

Characteristics and Regimens	Strength Training	Endurance Training	*P*
Sex (male/female)	11/11	9/8	
Age (y)	56.4±1.1	57.9±1.4	NS
Duration of diabetes (y)	8.83±3.5	9.2±1.71	NS
Treatment regimens			NS
Antidiabetic drug therapy			
Sulphonylurea	11	11	NS
Metformin*	15	13	NS
Insulin therapy	4	3	NS
Lipid-lowering drug therapy			
Statins†	8	7	NS
Antihypertensive drug therapy			
3 or more different antihypertensive medications	14	13	NS

NOTE. Values are n or mean ± SE.
Abbreviation: NS, not significant.
*Biguanide.
†A hyrdoxymethyl glutaryl coenzyme A reductase inhibitor.

All participants were told to continue their current medications during the study. Medications (especially sulphonylureas) were modified only to avoid hypoglycemia. All participants received specific recommendations to keep their energy intake unchanged during the 4-month training period.

The Ethics Committee at the Confraternitaet Hospital, Vienna, approved the study protocol. The purpose, nature, and potential risks of the study were explained to the participants before obtaining their written consent.

Training Program

We tried to define comparable training units for both groups. A unit is defined as an organizational unit for both training groups where training occurs. To do this, we took comparable training units of top athletes for each training group. A top weight-lift body builder, for example, does 30U per muscle group per week, whereas a top endurance athlete trains for 10 to 12 hours a week. For our study, we took 15% to 20% of these training units (repetitions by sets) for each group.

Endurance training. Systematic ET was performed on a cycle ergometer on 3 nonconsecutive days of the week. During the first 4 weeks, ET participants trained for 15 minutes per session, 3 times a week. Exercise sessions were increased by 5 minutes every 4 weeks. The total exercise time per week, excluding warmup and cool down, was 90 minutes during the last 4 weeks.

Heart rate (HR) was monitored continuously throughout the training period.[a] Based on the linear correlation between $\dot{V}O_2$ and heart rate, training was controlled by a heart rate according to 60% of $\dot{V}O_2$max. This was derived from ergometry by using the following formula[22]:

$$HR = HRrest + (HRmax - HRrest) \times 0.6 \pm 5 \text{ beats/min}$$

where HRrest was heart rate after a break of 5 minutes, in supine position.